HYPERTROPHIC PYLORIC STENOSIS IN THE INFANT.

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THE following case affords material for a critical consideration of several points of great significance in the evolution of the clinical history of pyloric stenosis in infants, what is commonly called congenital pyloric stenosis, but the term "congenital" implies a theory, and would be better discarded.

The child was the first-born of its parents, two young people who had always enjoyed excellent health. The child was born at full time, was of the average size, and in all respects was a plump, healthy-looking infant, a male. It was nursed by the mother, and for the first six weeks thrived exceedingly.

On the day on which he completed his sixth week of age, he began to put up a little now and again after a drink. The vomiting was sudden, and the material was ejected *en masse* with force, sometimes apparently part only of what he had taken, sometimes what seemed the whole, and sometimes there was put up after a meal what seemed the amount of two drinks. Day after day the tendency to put up, after a drink, a portion or all of the meal increased. He was at the time in the country, and I did not see him till ten days after the commencement. He was then still plump and healthy, but he had, his nurse said, lost considerably. At that time there was nothing else she had to note except the persistent ejection after a drink of a portion or all of the milk, always with the same explosive suddenness and force.

I stripped him naked and examined him carefully, and could find nothing of a definite character. To the local practitioner, however, I expressed the opinion that there was a suggestion of three nerve phenomena: (1) A sluggishness of pupils, (2) a faint rigidity of the muscles of the neck, (3) a tendency to turn the eyes downward. The suggestion was so slight that one could not insist on its presence, but later these symptoms became quite distinct.

The child, failing to improve, was brought back to town a

week later, nursing was discontinued, and he was fed by means of a spoon with a milk mixture in very small quantities. At this date he was eight weeks old exactly; he had lost considerably, though he was still not ill-nourished. The temperature in the bowel had been always normal, but on the sixteenth day from the commencement of the vomiting, it reached 100° F. in the rectum; the pulse was 105 and occasionally intermittent; the respiration was normal, and the pupils reacted slowly to changes of light.

A period followed, occupied by attempts to feed without inducing vomiting, by the use of very small quantities, frequently repeated, of milk mixtures peptonized of various dilutions, Carnrick's food for infants, white of egg in water, etc., being given each a fair trial, and in this way several days passed without much being lost. Thus on the twenty-second day of the illness nothing was vomited for twenty-four hours, twelve ounces of fluid having been given. In the interval between the sixteenth and twenty-second days an effort had been made to substitute rectal for gastric feeding wholly for a period, nothing but water being given by the mouth. But this had to be abandoned, the rectum proving fully as irritable as the stomach, very nearly, indeed quite, intolerant.

During this period, also, an analysis had twice been made of the gastric contents, obtained by the vomit being caught in a clean vessel.

The characters of the vomit were these: an extremely viscid, semigelatinous fluid, white in color, slightly frothy in appearance. It moved like a gelatinous mass in the glass vessel when it was shaken. It was very sour to smell and acid to litmus. The total acidity equalled 17 cubic centimetres decinormal solution to 100 of the filtered contents, and these 17 cubic centimetres were represented as follows:

		Cubic Centimetres.
Free HCl	=	0.00.
Acid salts	=	10.00.
Free organic acid	=	5.00.
Organic HCl	=	2.00.
		17.00.

A sample obtained a day or two later was so gelatinous that it would not filter. Because of the total absence of free HCl,

a mixture containing 0.2 per cent. HCl was given, and under its influence the vomit lost its remarkable viscosity.

Dilute peptonized milk seeming to be retained better than other foods, it was resumed in small quantities gradually increased, till two ounces in spoonfuls could be given within half an hour, and this repeated every two hours with little loss. Meanwhile, however, the temperature behaved in an erratic manner. On the twenty-fourth day, for instance, it was as follows: 4 A.M., 102°; 8 A.M., 102.3°; 12 noon, 100.4°; 6.30 P.M., 100.7°; 8 P.M., 99.3°. On the following day it was never above normal, and was taken in rectum every four hours.

On the twenty-sixth and following days it was as follows:

Twenty-sixth.	Twenty-seventh.	Twenty-eighth.	Twenty-ninth.	Thirtieth.
4 A. M..... 98.3°	100°	100.3°	99.4°	103.2°
8 A. M.....103°	98.4°	101.2°	99.4°	103.2°
12 M. .....101.3°	97.3°	100.3°	100°	101.3°
4 P. M.....101.2°	98.4°	100.2°	99.2°	99°
8 P. M..... 99.4°	98.4°	98.6°	98.4°	98°
10 P. M.....	.....	.....	102°	.....

From the sixteenth day of illness to the thirtieth day, the chief facts had been (1) the vomiting of more or less food, sometimes little, sometimes much, occurring from once to three or four times in the twenty-four hours, always with the same explosive force; (2) the erratic temperature; (3) elusive suggestions of nerve symptoms such as already noted. In appearance the child varied in an extraordinary way,—at one time being apathetic, pale, and pinched, with dark rings round the eyes, the eyes themselves dull and listless, with sluggish pupils; at other times he would appear fresh colored and bright, eyes alert, obviously taking notice, pupils freely mobile and expressive. An access of gastric irritability was usually preceded by the child becoming quiet, white, and haggard looking; then would follow the ejection of some food, after which his color and animation might return.

I have already commented on the irritability of rectum which made it impossible to substitute even for a season rectal for gastric feeding. The pharynx was equally irritable. In the course of the period under review repeated examinations were made of

chest and abdomen in general to assure oneself that nothing was being overlooked. If in such an examination any attempt was made with a spoon to depress the tongue, the stomach was immediately evacuated. The pharynx was always clean, but also always hyperæmic. Similarly, palpation over the epigastrium immediately caused vomiting, but up to the thirtieth day of illness no dilatation nor other abnormality had been made out, though repeatedly looked for.

During the whole of this period the child was kept lying on an ordinary single bed, from which he was lifted only for necessary purposes, and he was watched day and night. On the twenty-eighth day he was dull, apathetic, and quiet; his eyes showed a tendency to double external strabismus. His respiration, hitherto regular, assumed a Cheyne-Stokes character, in groups of three or four. The pulse at the time was 134 and the respirations 20 per minute. The rigidity of muscles of the back of the neck was quite obvious.

Towards the end of this period constipation began to be noticeable, what was passed from the bowel containing extremely little fæcal matter and being mainly dark, bile-stained mucus.

On the thirtieth day he was seen by Professor Stockman, when for the first time palpation over the epigastric region failed to induce vomiting. Vomiting had been so inevitable and immediate a sequel of any attempt to palpate the stomach, that as soon as Dr. Stockman began to palpate he was spontaneously warned by the mother of what would happen, and when it failed to happen, he was immediately informed by both nurse and mother, without any suggestion of mine, that an attempt to examine in this way had never before failed to provoke evacuation of the stomach. I lay stress upon this point because for the first time also bulging of the stomach was obvious, and for the first and only time gastric peristalsis was visible. In short, only when the stomach failed to empty itself quickly after a meal was there a suggestion of dilatation and of peristalsis, and this was not till the thirtieth day after vomiting had begun, when the child was, in other respects, also failing visibly. On this day, however, no tumor could be felt, but only once had a small quantity been ejected, and that only apparently because of castor oil, while sixteen ounces of fluid had been imbibed.

For a couple of days after this little nourishment was lost,

but a day later—the thirty-third of the illness—I detected on palpation a thickening in the region of the pylorus, and decided to have a surgical opinion. The pylorus could not be felt; the stomach seemed large; but when a No. 10 soft rubber catheter was passed, the stomach was found quite empty but for a little clear, viscid mucus. It was washed out with normal saline, and the returned fluid was quite free from odor, and contained only a plug or two of thick mucus. The child, however, was looking extremely ill; temperature in rectum was 104° F., and pulse 148; the surgeon did not think there was the smallest hope of an operation having a successful issue.

The following evening the child had a convulsive seizure, which recurred within an hour, and continued to recur at intervals of an hour and sometimes less for the next twenty-two hours, till operation was performed. The operation was undertaken in response to the request of the mother, who had done a large part of the nursing, had followed with great intelligence the various phases of the illness, and realized how hopeless the situation had become unless surgical interference could find some way of escape.

Contrary to expectation, the child bore the operation without difficulty, and took the chloroform easily; in fact, breathing and pulse improved under it.

The whole gastric wall was found to be uniformly hypertrophied, though dilatation was not marked, and the extreme pyloric thickening made clear the true nature of the disease. A gastro-enterostomy was performed, but under great difficulty, the duodenal wall being extremely thin and the lumen small. The gastric mucosa was very hyperæmic and highly sensitive, the act of introducing a small gauze plug through the wound in the gastric wall inducing a violent pharyngeal reflex, repeated when the gauze was withdrawn. The internal surface of the stomach wall was coated with a thick layer of transparent mucus, of high viscosity, to which I was subsequently disposed to attribute, in part at least, the failure of the operation.

The child survived the operation fourteen hours. Immediately after the operation the temperature stood at 96° F. in the rectum, but it rose to 99° F., and was at 98.8° F. shortly before death.

Convulsive attacks remained in abeyance for six hours after

operation, but then recurred repeatedly, and death was preceded by a severe one. Vomiting also was frequent in the interval between operation and death. A post-mortem was not permitted, but I was allowed to remove the stomach through the operation wound, and I removed it without disturbing the short circuit.

When a glass tube had been tied into the cardiac end, and the stomach was filled with water, it remained full, apparently not a single drop leaking through the pylorus. The photograph (Fig. 1) gives a very accurate view of the appearance of the organ when so distended. Fig. 2 shows the stomach opened up longitudinally, and reveals the distribution of the hypertrophy; the extremely narrow channel through the pylorus left patent by the hypertrophy was occluded by the rugæ and the dense secretion, and the outlet the operation meant to provide was similarly obstructed by viscid mucus. The operation, therefore, had secured only a brief relaxation of the extreme irritability of the stomach, but no actual outlet had been obtained. Fig. 3 is the photograph (× 6 diameters) of the hypertrophied pylorus, the hypertrophied portion passing into stomach on the right and duodenum on the left, the mucous surface being uppermost. An X on Fig. 2 shows the part from which this section was removed. The section was stained in picrocarmine, and Fig. 4 is a drawing of the left hand extremity of Fig. 3, where pylorus projects into duodenum.

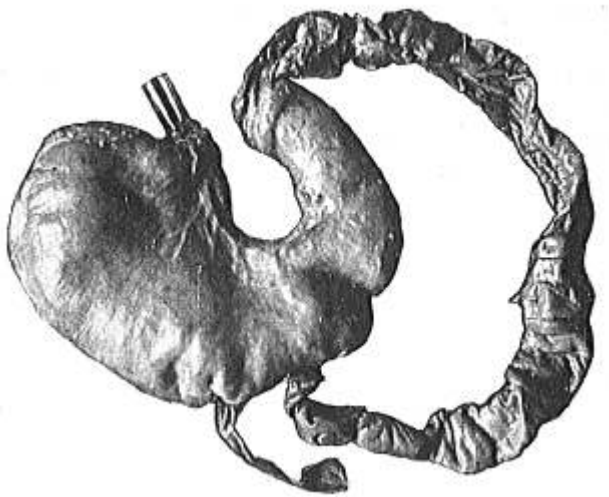

FIG. 1.—Infantile pyloric stenosis. Appearance of stomach removed post-mortem and distended with water.

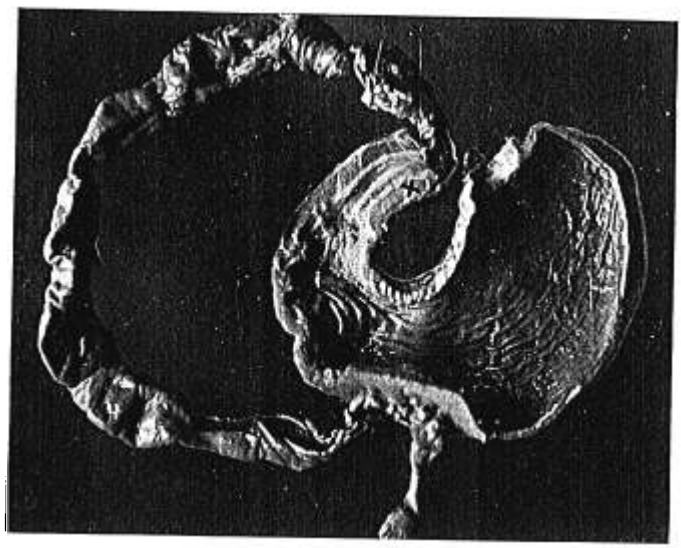

FIG. 2.—Infantile pyloric stenosis: Longitudinal section. Congenital section. Note hypertrophic thickening of wall of pylorus indicated at ×, from which point tissue shown in Figs. 3 and 4 was taken.

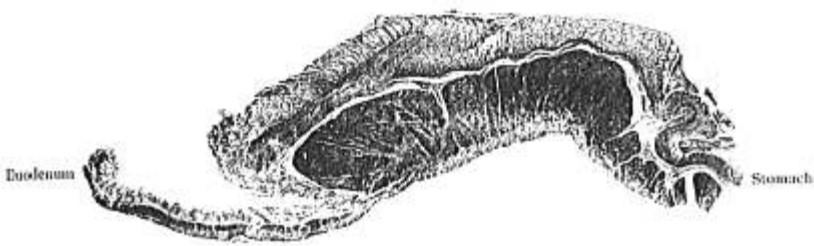

FIG. 3.—Congenital hypertrophy of wall of pylorus. × 6 diameters.

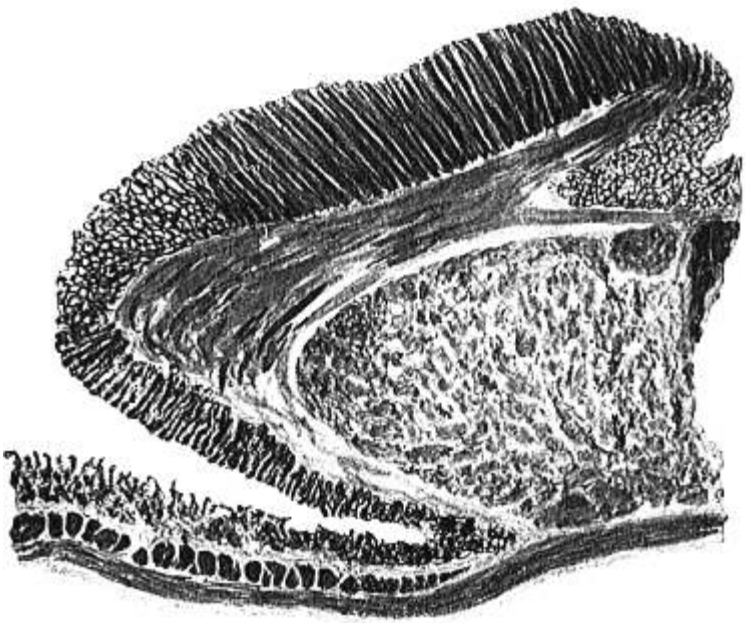

FIG. 4.—Gastroduodenal junction. × 12 diameters.